Correlation between Surface and Intracardiac Electrocardiogram in a Patient with Inappropriate Defibrillation Shocks Due to Hyperkalemia

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Abstract

A 39-year-old man received implantable cardioverter defibrillator (ICD) shocks during sinus rhythm, triggered by an increase in amplitude and oversensing of intracardiac T waves, caused by hyperkalemia. After treatment of hyperkalemia, the T wave morphology normalized, and oversensing and inappropriate ICD shocks were eliminated. Alteration of the intracardiac electrogram was well correlated to the surface electrocardiogram (ECG) changes. Intracardiac T waves can be altered by hyperkalemia and it seems that this alteration can be estimated by surface ECG analysis.

Key words: T wave oversensing, hyperkalemia, inappropriate defibrillator shock, implantable cardioverter defibrillator

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Introduction

Inappropriate implantable cardioverter defibrillator (ICD) shocks due to T wave oversensing is typically difficult to predict from surface electrocardiogram (ECG) analysis, but it can be life-threatening when delivered during the vulnerable period of the cardiac cycle (1). We recently observed a patient in whom inappropriate ICD shocks were delivered because of oversensing of intracardiac T waves accentuated by hyperkalemia. In this patient, alteration of the local electrogram was well correlated with the surface ECG changes.

Case Report

A 39-year-old man was admitted to our hospital for management of ICD shocks delivered during sinus rhythm. He had suffered, 18 months earlier, two syncpe episodes and ventricular tachycardia (VT) at the rate of 210 bpm was documented in one of the episodes. His 12-lead ECG at rest was normal and Brugada-type ECG abnormalities were not observed in repetitive ECG recordings. Structural heart diseases were not apparent by conventional cardiac examinations. Coronary angiograms were normal and the left ventriculogram revealed a 69% ejection fraction. During the electrophysiological study, clinical VT could not be reproduced but ventricular fibrillation and non-clinical polymorphic VT were reproducibly induced by programmed electrical stimulation. Catheter ablation was not attempted because an arrhythmogenic area of the induced ventricular tachyarrhythmias and origin of the clinical VT could not be determined. Since the most likely cause of his syncpe episodes was considered to be ventricular tachyarrhythmia, a Gem II VR single chamber pulse generator (Medtronic Inc., Minneapolis, MN) was implanted and connected to a 6,945-65 RV lead (Medtronic). Detection of the ventricular tachyarrhythmias was programmed in the ICD device as follows. VF was programmed as the cycle length (CL) of less than 300 ms, fast VT was set as the CL between 270 and 300 ms, and VT was defined as the CL between 320 and 300 ms. For the treatment, a set of up to six shocks at maximum energy (30 J) was programmed for VF, and a set of up to

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five shocks (30 J) following the initial anti-tachycardia pacing was set for fast VT and VT. After his discharge from the hospital on antihypertensive therapy with candesartan cilexetil, 4 mg/day, the patient received several appropriate ICD shocks and antitachycardia pacing for recurrent monomorphic VTs at the CL of 260-320 ms. Serum potassium level at the time was relatively low (3.4-3.6 mEq/L) and we prescribed additional drugs of atenolol, 50 mg/day, and potassium chloride, 2.4 g/day. This treatment decreased the VT recurrence rate, and serum potassium remained stable between 3.9 and 4.5 mEq/L.

A few days before this hospitalization, the patient developed frequent vomiting and diarrhea attributed to “common cold”. Despite loss of appetite, he continued to take his medications and, shortly thereafter, received several ICD shocks preceded by no symptom. The 12-lead ECG at the time of admission showed sinus rhythm, with accentuated and peaked T waves, particularly in leads V2-V4 (Fig. 1). Laboratory tests revealed serum potassium at 8.3 mEq/L, creatinine at 2.00 mg/dL, and blood urea nitrogen at 33 mg/dL. Interrogation of the ICD revealed that the cause of inappropriate shock delivery was T wave oversensing due to an increase in the intracardiac T wave amplitude (Figs. 2, 3). At the time of device implantation, the peak-to-peak amplitude of the intracardiac R and T waves were 13.0 and 2.5 mV, respectively, and T wave oversensing was not observed at a sensing threshold of 0.3 mV. While the intracardiac R and T waves remained stable during follow-up, the peak-to-peak T wave amplitude at the time of admission had increased to 4.5 mV, and its slope was steeper (Fig. 3). The R wave morphology, however, had remained stable, and the ICD counted both R and T waves at the sensing threshold of 0.3 mV, which satisfied the ventricular fibrillation criterion. His ventricular pacing threshold was stable (2.0 V×0.2 ms) before and during the episode of transient hyperkalemia. All medications were immediately discontinued, 5% glucose with insulin and calcium gluconate were administered intravenously, and the serum potassium normalized to 4.6 mEq/L within 1 day. Likewise, the intracardiac and surface ECG T wave morphology normalized at the same time. After treatment, the T wave morphology remained stable during changes in position and during treadmill exercise test. He was discharged from our hospital with the prescription of candesartan cilexetil, 4 mg/day, atenolol, 50 mg/day, and potassium chloride, 1.2 g/day, and neither T wave oversensing nor inappropriate ICD discharge have recurred over a 16-month follow-up.

**Discussion**

The manifestations of hyperkalemia on surface ECG include a short QT interval and tall T wave, due to an increase in net outward currents by augmentation of I_k1 and I_kr, which hastens myocardial repolarization and creates a large voltage gradient between ventricular mid-myocardium and epicardium during phases 3 and 4 of repolarization (2). Although it increases the amplitude of the T wave on surface ECG, hyperkalemia is a rare cause of T wave oversensing and inappropriate ICD discharge (3, 4). In the present patient, hyperkalemia was apparently induced by the combined effects of medications and dehydration during a minor illness. The T wave augmentation on surface ECG was consistent with hyperkalemia, which was observed only during the period of hyperkalemia.

Similar T wave oversensing and inappropriate ICD discharges have been observed in patients with cardiac sarcoidosis, idiopathic cardiomyopathy, Brugada syndrome and congenital long QT syndrome (5-7). In those patients, the
inappropriate ICD shock following T wave oversensing during sinus rhythm.}

**Figure 2.** Delivery of inappropriate ICD shock following T wave oversensing during sinus rhythm.

**Figure 3.** Intracardiac electrocardiograms recorded from the tip and ring electrodes of the ICD lead. A and B: Before the onset of inappropriate shocks, the intracardiac R and T waves were stable and no T wave oversensing was detected. C: At the time of admission, the T wave amplitude was increased and peaked, while the R wave was stable. D: After treatment of hyperkalemia, the T wave configuration returned to baseline.

intracardiac R wave amplitude was decreased while the T wave amplitude was increased. These changes would be attributed to alteration in the activity of cardiac sarcoidosis or progressive myocardial injury in a certain area but not the whole ventricle. In Brugada syndrome, primary arrhythmogenic substrate is considered to be located in the right ventricle (6). Therefore, alteration of the local electrograms in these patients is not always detected on the surface ECG. In contrast, hyperkalemia alters the entire ventricular repolarization process, such that a close correlation is expected between changes in intracardiac electrograms and surface ECG, as was observed in the present patient. Similar results may occur in patients with congenital long QT syndrome. However, in a literature search, we did not find any detailed report of a correlation between intracardiac electrograms and surface ECG at the time of T-wave oversensing in congenital long QT syndrome.

Although it seems rare, hyperkalemia can augment local T wave amplitude large enough to be detected by ICD. A recent study showed that both appropriate and inappropriate ICD shocks were associated with an increase in the risk of total death in patients with left ventricular dysfunction (8). Therefore, physicians need to pay attention to the serum potassium level and T wave morphology on surface ECG during the follow-up of ICD recipients, especially those who suffer from renal dysfunction and are treated by angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, because this phenomenon may trigger inappropriate ICD deliveries and subsequently induce cardiac dysfunction.
References


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